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APPLICATION NO.	FILING DATE	FIRST NAMED INVENTOR	ATTORNEY DOCKET NO.	CONFIRMATION NO.
09/955,174	09/19/2001	William G. Kerr	USF-T150CX	9411

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EXAMINER

ZARA, JANE J

ART UNIT	PAPER NUMBER
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1635

DATE MAILED: 12/29/2005

Please find below and/or attached an Office communication concerning this application or proceeding.

Office Action Summary

Application No.

09/955,174

Applicant(s)

KERR, WILLIAM G.

Examiner

Jane Zara

Art Unit

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-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE 3 MONTH(S) OR THIRTY (30) DAYS, WHICHEVER IS LONGER, FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133). Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

- 1) ☒ Responsive to communication(s) filed on 07 October 2005.
- 2a) ☒ This action is **FINAL**. 2b) ☐ This action is non-final.
- 3) ☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

- 4) ☒ Claim(s) 38-89 is/are pending in the application.
- 4a) Of the above claim(s) _____ is/are withdrawn from consideration.
- 5) ☐ Claim(s) _____ is/are allowed.
- 6) ☒ Claim(s) 38-89 is/are rejected.
- 7) ☐ Claim(s) _____ is/are objected to.
- 8) ☐ Claim(s) _____ are subject to restriction and/or election requirement.

Application Papers

- 9) ☐ The specification is objected to by the Examiner.
- 10) ☒ The drawing(s) filed on 19 September 2001 is/are: a) ☒ accepted or b) ☐ objected to by the Examiner.
Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).
Replacement drawing sheet(s) including the correction is required if the drawing(s) is objected to. See 37 CFR 1.121(d).
- 11) ☐ The oath or declaration is objected to by the Examiner. Note the attached Office Action or form PTO-152.

Priority under 35 U.S.C. § 119

- 12) ☐ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).
- a) ☐ All b) ☐ Some * c) ☐ None of:
- ☐ Certified copies of the priority documents have been received.
 - ☐ Certified copies of the priority documents have been received in Application No. _____.
 - ☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

* See the attached detailed Office action for a list of the certified copies not received.

Attachment(s)

- 1) ☒ Notice of References Cited (PTO-892)
- 2) ☐ Notice of Draftsperson's Patent Drawing Review (PTO-948)
- 3) ☒ Information Disclosure Statement(s) (PTO-1449 or PTO/SB/08)
Paper No(s)/Mail Date 5/02, 2/03, 3/03, 2/05, 4/05, 10/05
- 4) ☐ Interview Summary (PTO-413)
Paper No(s)/Mail Date. _____
- 5) ☐ Notice of Informal Patent Application (PTO-152)
- 6) ☐ Other: _____

DETAILED ACTION

This Office action is in response to the communication filed 10-7-05.

Claims 38-89 are pending in the instant application.

The text of those sections of Title 35, U.S. Code not included in this action can be found in a prior Office action.

The declarations under 37 CFR 1.132 filed 7-21-04 and 2-9-05 are insufficient to overcome the rejection of claims 38-89 based upon 35 U.S.C. 112, first paragraph, as set forth in the last Office action because for the reasons set forth below.

Response to Arguments and Amendments

Withdrawn Rejections

Any rejections not repeated in this Office action are hereby withdrawn.

Maintained Rejections and New Rejections Necessitated by Amendments

Claims 38-89 are rejected under 35 U.S.C. 112, first paragraph, as failing to comply with the written description requirement for the reasons of record set forth the Office action mailed 5-5-05 and for the reasons set forth below. This is both a new matter rejection and a written description rejection, and the arguments for maintaining both grounds of rejection are set forth below.

The claims are drawn to compositions and methods comprising the administration of an interfering RNA (RNAi) specific for SHIP-1 mRNA that is present in human or mouse hematopoietic cells, and comprising a nucleic acid molecule that

hybridizes in vitro under conditions of stringency with human or mouse SHIP-1 mRNA or hybridizes in vivo with SHIP-1 mRNA present in mouse or human hematopoietic cells and reduces SHIP-1 function in human or mouse, suppresses graft-versus-host disease in a mouse or human, and suppresses transplant rejection in a human or mouse.

Applicant's arguments filed 10-7-05 and declarations filed 7-21-04 and 2-9-05 have been fully considered but they are not persuasive. Applicant argues generally that the original disclosure provides adequate support and adequate written description for the RNAi molecules and methods claimed. Applicant argues that mRNA sequences of mouse and human SHIP-1 have been publicly available since the late 1990's and nucleotide homology has been provided for human and mouse mRNA sequences. Applicant is correct that nucleotide sequences of the target genes encoding human and mouse SHIP-1 have been reported in the 1990s, but the publication of target gene sequences is not representative of adequate description of RNAi sequences, which sequences were adequately described and in one's possess at the time of filing, which RNAi sequences bind to and reduce SHIP-1 function in vivo, and suppress graft-versus-host disease and transplant rejection. In addition, various splice isoforms and sequence variants have been reported for mouse or human SHIP-1 (see e.g. Liu et al., Genomics, Vol. 39, pages 109-112, 1997, abstract and introduction on p. 109 and Fig. 1 on p. 110; see also Wolf et al., Genomics, Vol. 69, pages 104-112, 2000, esp. the abstract and Fig. 4 on p. 108). The specification and claims do not adequately describe the concise structural features that distinguish structures within the claimed genus from those without (e.g. the exact nucleotide sequences or a representative number of RNAi

molecules of the generic RNAi structures claimed, that specifically bind and inhibit SHIP-1 function in vivo, and which suppress graft-versus-host disease and transplant rejection). One of skill in the art would reasonably conclude that the disclosure fails to provide a representative number of species of the inhibitory molecules claimed, encompassing the genus comprising RNAi specific for SHIP-1 mRNA present in human or mouse hematopoietic cells or comprising any nucleic acid molecule (and of any size) that hybridizes in vitro under conditions of stringency with human or mouse SHIP-1 mRNA or hybridizes in vivo with SHIP-1 mRNA present in mouse or human hematopoietic cells and provides the treatment effects claimed.

Applicants argue that the subject specification provides sufficient information regarding the genus of SHIP-1 mRNA as well as interfering RNA specific thereto. Contrary to Applicant's assertions, the original disclosure (filed 9-19-01) makes no mention of interfering RNA. The declaration filed later in prosecution, on 7-21-04, provides a disclosure of experiments in which SiRNA (*a.k.a.* interfering RNA or RNAi) molecules #1, 2, 3 and 4, or a combination or subcombination of them, were found to successfully inhibit the expression of SHIP-1 in vitro and in vivo. The original application, however, does not provide disclosure of these SiRNA molecules, nor of these experiments, nor of any mention of interfering RNA molecules in general. Applicants provide a supplemental IDS (filed 10-7-05) with various references teaching RNAi molecules and their use in RNAi-mediated gene suppression in mammalian cells. The existence of these publications, however, is no substitute for the original disclosure of this invention in the subject patent application.

Applicant also argues that, while the predictability that any single interfering RNA molecule will be effective in gene silencing is not necessarily high, the probability of identifying an individual functional interfering RNA molecule among candidates is very high... Contrary to Applicant's assertions, the eventual identification of molecules among a myriad of candidate possibilities is no substitute for the requirement of having in one's possession, at the time of filing, a representative number of species for such a broad genus claimed, as in the instant case, for the genus comprising RNAi specific for SHIP-1 mRNA present in human or mouse hematopoietic cells, or that hybridize in vitro under conditions of stringency with human or mouse SHIP-1 mRNA, or hybridize in vivo with SHIP-1 mRNA present in mouse or human hematopoietic cells, and reduce SHIP-1 function in human or mouse, or suppress graft-versus-host disease or transplant rejection in a mouse or human. For these reasons, the instant rejection is maintained.

Claims 38-66 and 74-89 are rejected under 35 U.S.C. 112, first paragraph, for lacking enablement over the scope claimed for the reasons of record set forth the Office action mailed 5-5-05 and for the reasons set forth below.

The claims are drawn to methods for interfering and reducing SHIP-1 function in a human or mouse, for suppressing transplant rejection in a human or mouse, and for suppressing graft versus host disease (GVHD) in a human or mouse comprising the administration, in vivo or ex vivo, of an RNAi specific for SHIP-1 mRNA present in human or mouse hematopoietic cells, or which RNAi hybridizes in vitro under conditions

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of stringency with human or mouse SHIP-1 mRNA or hybridizes in vivo with SHIP-1 mRNA present in mouse or human hematopoietic cells.

The instant disclosure, while being enabling for a method of suppressing the rejection of an allogeneic bone marrow graft from BALB/C mice in SHIP-/- mice or abrogating GVHD disease in SHIP-/- mice that were transplanted with whole bone marrow from BALB/C mice, thereby enhancing SHIP-/- mice survival, and while being enabling for the in vivo inhibition of SHIP-1 expression in mice using the RNAi sequences #1, #4 and the mouse antisense vector muSHIPshRNA provided in the declarations by Dr. Kerr, filed 7-21-04 and 2-9-05, does not reasonably provide enablement for inhibiting SHIP-1 in vivo comprising the administration of *any* RNAi specific for SHIP-1 mRNA present in human or mouse hematopoietic cells, or comprising the administration in vivo or ex vivo of *any* nucleic acid molecule that hybridizes in vitro under conditions of stringency with human or mouse SHIP-1 mRNA or that hybridizes in vivo with SHIP-1 mRNA present in mouse or human hematopoietic cells, nor of suppressing a transplant rejection in any patient, or treating graft versus host disease (GVHD) in any patient comprising the administration of any interfering RNA specific for SHIP mouse or human mRNA.

Applicant's arguments filed 10-7-05 and declarations filed 7-21-04 and 2-9-05 have been fully considered but they are not persuasive. Applicant argues that the instant invention is enabled for the full scope claimed because one of ordinary skill in the art would be able to make and use the invention without undue experimentation. Applicant argue that the specification teaches at pages 11 and 13 that inhibitors of SHIP

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can be administered to cells ex vivo. Contrary to Applicant's assertions, the mere recitation of the existence of a preferred technique is not enabling for that technique. Ex vivo enablement requires the removal of the cells from the body, treatment of those cells with a representative number of species of the genus claimed, and subsequently providing the treatment effects claimed following a return of the treated cells to the body of the organism. This requires experimentation beyond that provided in the instant disclosure, and for the breadth of the scope claimed, whereby a representative number of SiRNAs have successfully reduced SHIP-1 function in vivo, and whereby transplant rejection and graft versus host disease (GVHD) have been suppressed following in vivo and/or ex vivo administration of the genus claimed.

Applicant argues that the issues raised in the previous enablement rejection mailed 5-5-05 and which pertain to the challenges of delivery are inapplicable to the compositions claimed in the subject application, especially as they pertain to ex vivo delivery of RNAi. Applicant also argues that the proper standard for compliance with the enablement standard is not absolute predictability but objective enablement. Applicant is correct that the challenges of delivery of oligonucleotides is diminished by ex vivo administration and treatment of cells, but no evidence has been provided in the instant disclosure for the ex vivo treatment of cells with RNAi, and further whereby treated cells are delivered to a subject and treatment effects are provided. Applicants have not provided guidance toward a method of suppressing a transplant rejection in a patient, or treating graft versus host disease (GVHD) in a patient comprising the administration of an interfering RNA specific for SHIP mRNA. The specification teaches

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the suppression of the rejection of an allogeneic bone marrow graft from BALB/C mice in SHIP-/- mice, as well as the abrogation of GVHD disease in SHIP-/- mice that were transplanted with whole bone marrow from BALB/C mice, whereby SHIP-/- mice survival was enhanced. The declarations filed 7-21-04 and 2-9-05 teach the in vivo inhibition of SHIP-1 following the co-administration of RNAi sequences #1, #4, or following the administration of the mouse antisense vector muSHIPshRNA. The ability of co-administered RNAi's, or of the mouse antisense vector muSHIPshRNA to target and successfully inhibit expression of the target gene encoding SHIP1 in a mouse model, and provide for an observed increase in Mac+Gr1 monocytes and circulating Mac1+GR1+cells (myeloid suppressor cells) is not representative of providing other treatment effects including altering NK function, suppressing transplant rejection in any patient, or treating graft versus host disease (GVHD) in any mammal following administration of a representative number of RNAi, or following administration of the mouse antisense vector muSHIPshRNA. Nor is it representative or correlative of the ability to achieve in vivo SHIP-1 inhibition of expression or subsequent treatment effects comprising the administration of any RNAi specific for SHIP-1 mRNA present in human or mouse hematopoietic cells or comprising any nucleic acid molecule (and of any size) that hybridizes in vitro under conditions of stringency with human or mouse SHIP-1 mRNA or hybridizes in vivo with SHIP-1 mRNA present in mouse or human hematopoietic cells. One skilled in the art would not accept on its face the examples given in the specification of the enhanced survival and reduction of transplant rejection in a mouse model using SHIP-/- mice as being correlative or representative of the

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successful inhibition of expression of SHIP in vivo using interfering RNA specific for SHIP mRNA, and further whereby treatment effects are provided for transplant rejection or graft versus host disease (GVHD) in a patient in view of the lack of guidance in the specification and known unpredictability associated with the ability to predict the efficacy of interfering RNA in inhibiting the expression of SHIP in a mouse or human in treating, suppression transplant rejection or graft versus host disease (GVHD) in a patient following administration by any route of the claimed RNAi oligonucleotides. The specification as filed fails to provide any particular guidance which resolves the known unpredictability in the art associated with in vivo or ex vivo delivery and treatment effects provided by RNAi administered, and specifically regarding the broad genus of compounds and methods claimed, which treatment methods comprise methods of suppressing graft versus host disease and transplant rejections in a subject.

Applicant argues that the references cited in the prior enablement rejection (e.g. of Branch, Crooke, Agrawal, Chirila and Peracchi cited in the 5-5-05 Office action) are not relevant in their discussion of the obstacles regarding limited oligonucleotide delivery to target cells in vivo because they do not consider interfering RNA. Applicant argues further that RNAi is distinguishable from antisense and ribozymes because less RNAi are required for effective inhibition of target genes. Applicant is correct that investigators have reported that lower concentrations of RNAi molecules are likely needed for effective inhibition in a target cells compared to antisense or ribozyme molecules (see e.g. Fire (USPN 6,506,559, at col. 3). However, contrary to Applicant's assertions, despite the supposition that lower concentrations of RNAi molecules are

likely required in target cells for effective target gene inhibition in comparison to antisense or ribozymes, in vivo efficacy of RNAi still depends on the effective delivery of threshold concentrations of these oligonucleotides sufficient to silence the target gene in target cells harboring the SHIP-1 target gene, and in vivo delivery of oligonucleotides, whether they be antisense, ribozymes or RNAi molecules, is generally a highly unpredictable endeavor at the current time (see e.g. Caplen for a recent review article concerning RNAi as an effective gene therapy tool (Caplen, N.J., Expert Opinion Biol. Ther., Vol. 3, No. 4, pages 575-586, 2003, esp. the bridging paragraph, at pp. 577-8: "While most siRNAs are effective in inducing some degree of gene silencing, there are wide ranges in the efficacy of individual siRNAs against sequences within the same gene, and some siRNAs show limited or no ability to mediate RNAi. It is currently unclear what specific parameters determine the effectiveness of a given siRNA and, thus, why some sequences may be better targets than others." See Caplen at p. 581: Many of the problems associated with developing RNAi as an effective therapeutic are the same as encountered with previous gene therapy approaches. The key issues of delivering nucleic acids to the required tissue and cell type, while ensuring an appropriate level of efficacy with minimum toxicity induced by the vector system have been problems the gene therapy field has struggled with for over a decade now.").

Claim Rejections - 35 USC § 103

The following is a quotation of 35 U.S.C. 103(a) which forms the basis for all obviousness rejections set forth in this Office action:

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(a) A patent may not be obtained though the invention is not identically disclosed or described as set forth in section 102 of this title, if the differences between the subject matter sought to be patented and the prior art are such that the subject matter as a whole would have been obvious at the time the invention was made to a person having ordinary skill in the art to which said subject matter pertains. Patentability shall not be negated by the manner in which the invention was made.

This application currently names joint inventors. In considering patentability of the claims under 35 U.S.C. 103(a), the examiner presumes that the subject matter of the various claims was commonly owned at the time any inventions covered therein were made absent any evidence to the contrary. Applicant is advised of the obligation under 37 CFR 1.56 to point out the inventor and invention dates of each claim that was not commonly owned at the time a later invention was made in order for the examiner to consider the applicability of 35 U.S.C. 103(c) and potential 35 U.S.C. 102(e), (f) or (g) prior art under 35 U.S.C. 103(a).

Claims 67-73 are rejected under 35 U.S.C. 103(a) as being unpatentable over Damen et al and Ware, the combination further in view of Fire and Gill et al.

The claims are drawn to compositions comprising RNAi specific for human or mouse SHIP-1 mRNA present in hematopoietic cells, and plasmid or viral vectors comprising RNAi, and which vectors are complexed with liposomes, and which compositions further comprises a pharmaceutically acceptable carrier.

Damen et al (Proc. Natl. Acad. Sci. USA, Vol. 93, pages 1689-1693, 1996) teach the cloning and characterization of mouse SHIP-1 obtained from hematopoietic cells, the nucleotide sequence encoding mouse SHIP-1, and its role as a signal transduction intermediate and its potential involvement in the modulation of Ras and inositol signaling pathways (see the abstract and introduction on p. 1689; fig. 2 on p. 1692; text on pp. 1691-2).

Ware et al (Blood, Vol. 88, No. 8, pages 2833-2840, 1996) teach the cloning and characterization of human SHIP-1, its nucleotide sequence, and its potential role in regulating receptor mediated signaling processes (abstract and introduction on p. 2833; fig. 2 on p. 2836).

The primary references of Damen and Ware do not teach compositions comprising RNAi that target and inhibit SHIP-1, nor do they teach plasmid or viral vectors comprising RNAi, nor vectors complexed with liposomes

Fire et al (USPN 6,506,559) teach RNAi molecules that target and inhibit the expression of a target gene of known sequence. Fire also teaches compositions comprising these RNAi molecules and a pharmaceutically acceptable carrier, and which RNAi is optionally in an expression vector and in a composition comprising a formulation for enhancing target cell delivery (see esp. the abstract; col. 4-9; col. 11-12; claims 1-3, 4, 6, 10, 12, 21).

Gill et al (USPN 5,804,412) teach compositions comprising viral or plasmid vectors comprising inhibitory molecules including antisense, and which compositions further comprise vectors complexed with liposomes (see col. 11, lines 29-52; col. 14, line 3-col. 15, line 10).

It would have been obvious to one of ordinary skill in the art to provide compositions comprising RNAi molecules that target and inhibit the expression of SHIP-1 in vitro. One of ordinary skill in the art would have been motivated to provide such compositions in order to use RNAi molecules to target and inhibit the expression of SHIP-1 in vitro because Ware et al and Damen both teach the role of SHIP-1 in the

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modulation of Ras and inositol signaling pathways, and its potential role in such cellular processes is feasibly investigated by inhibiting its expression in vitro using the routine method of oligonucleotide based inhibition, including by antisense or RNAi inhibition. It would have been obvious to design RNAi molecules for inhibiting expression of SHIP-1 in vitro because Fire teaches this routine technique and its enhanced effectiveness in target gene inhibition using these RNAi molecules compared to antisense in target gene inhibition (see e.g. col. 1-3 and Table 1 in col. 22-24 of Fire). Using the teachings of Fire, it would have been routine experimentation to design compositions comprising various RNAi molecules to inhibit the expression of the mouse or human SHIP-1 molecule because the nucleotide sequences of mouse and human SHIP-1 have been taught previously by Damen and Ware, and Fire teach the routine technique of designing RNAi to target and inhibit the expression of target genes of known sequence. One of ordinary skill in the art would have been motivated to produce the compositions comprising RNAi which target and inhibit the expression human and mouse SHIP-1 in order to study its role in the cellular processes of signaling because Damen and Ware teach the potential role of SHIP-1 in these important cellular processes whose aberrations are being investigated in various pathological conditions or diseases. One of ordinary skill in the art would have been motivated to provide compositions further comprising either viral or plasmid vectors comprising RNAi molecules, and optionally further comprising liposomes because these compositions are routinely used to enhance delivery and subsequent expression of inhibitory oligonucleotides to target cells in vitro for targeting and inhibition of a desired target gene sequence. For these

reasons, the instant invention would have been obvious to one of ordinary skill in the art at the time the invention was made.

Double Patenting

The nonstatutory double patenting rejection is based on a judicially created doctrine grounded in public policy (a policy reflected in the statute) so as to prevent the unjustified or improper timewise extension of the "right to exclude" granted by a patent and to prevent possible harassment by multiple assignees. A nonstatutory obviousness-type double patenting rejection is appropriate where the conflicting claims are not identical, but at least one examined application claim is not patentably distinct from the reference claim(s) because the examined application claim is either anticipated by, or would have been obvious over, the reference claim(s). See, e.g., *In re Berg*, 140 F.3d 1428, 46 USPQ2d 1226 (Fed. Cir. 1998); *In re Goodman*, 11 F.3d 1046, 29 USPQ2d 2010 (Fed. Cir. 1993); *In re Longi*, 759 F.2d 887, 225 USPQ 645 (Fed. Cir. 1985); *In re Van Ornum*, 686 F.2d 937, 214 USPQ 761 (CCPA 1982); *In re Vogel*, 422 F.2d 438, 164 USPQ 619 (CCPA 1970); and *In re Thorington*, 418 F.2d 528, 163 USPQ 644 (CCPA 1969).

A timely filed terminal disclaimer in compliance with 37 CFR 1.321(c) or 1.321(d) may be used to overcome an actual or provisional rejection based on a nonstatutory double patenting ground provided the conflicting application or patent either is shown to be commonly owned with this application, or claims an invention made as a result of activities undertaken within the scope of a joint research agreement.

Effective January 1, 1994, a registered attorney or agent of record may sign a terminal disclaimer. A terminal disclaimer signed by the assignee must fully comply with 37 CFR 3.73(b).

Claims 38-66 and 74-89 are provisionally rejected on the ground of nonstatutory obviousness-type double patenting as being unpatentable over claims 12, 13, 22-24, 33-40 of copending Application No. 10/097,101. Although the conflicting claims are not identical, they are not patentably distinct from each other because claims 38-66 and 74-89 are drawn to methods of reducing transplantation rejection in a human or mouse comprising the administration of RNAi specific for SHIP mRNA and claims 12, 13, 22-24, 33-40 of copending Application No. 10/097,101 are drawn to methods of reducing

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allogeneic transplantation comprising the administration of RNAi specific for mammalian SHIP, including human and mouse SHIP mRNA.

This is a provisional obviousness-type double patenting rejection because the conflicting claims have not in fact been patented.

Conclusion

Applicant's amendment necessitated the new ground(s) of rejection presented in this Office action. Accordingly, **THIS ACTION IS MADE FINAL**. See MPEP § 706.07(a). Applicant is reminded of the extension of time policy as set forth in 37 CFR 1.136(a).

A shortened statutory period for reply to this final action is set to expire **THREE MONTHS** from the mailing date of this action. In the event a first reply is filed within **TWO MONTHS** of the mailing date of this final action and the advisory action is not mailed until after the end of the **THREE-MONTH** shortened statutory period, then the shortened statutory period will expire on the date the advisory action is mailed, and any extension fee pursuant to 37 CFR 1.136(a) will be calculated from the mailing date of the advisory action. In no event, however, will the statutory period for reply expire later than **SIX MONTHS** from the date of this final action.

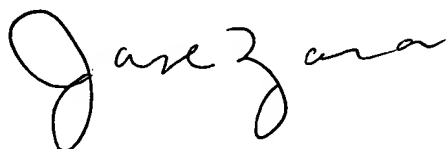
Certain papers related to this application may be submitted to Art Unit 1635 by facsimile transmission. The faxing of such papers must conform with the notices published in the Official Gazette, 1156 OG 61 (November 16, 1993) and 1157 OG 94 (December 28, 1993) (see 37 C.F.R. 1.6(d)). The official fax telephone number for the

Group is **571-273-8300**. NOTE: If Applicant *does* submit a paper by fax, the original signed copy should be retained by applicant or applicant's representative. NO DUPLICATE COPIES SHOULD BE SUBMITTED so as to avoid the processing of duplicate papers in the Office.

Any inquiry concerning this communication or earlier communications from the examiner should be directed to **Jane Zara** whose telephone number is **(571) 272-0765**. If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Andrew Wang, can be reached on (571) 272-0811. Any inquiry regarding this application should be directed to the patent analyst, Katrina Turner, whose telephone number is (571) 272-0564. Any inquiry of a general nature or relating to the status of this application should be directed to the Group receptionist whose telephone number is (703) 308-0196.

Information regarding the status of an application may be obtained from the Patent Application Information Retrieval (PAIR) system. Status information for published applications may be obtained from either Private PAIR or Public PAIR. Status information for unpublished applications is available through Private PAIR only. For more information about the PAIR system, see <http://pair-direct.uspto.gov>. Should you have questions on access to the Private PAIR system, contact the Electronic Business Center (EBC) at 866-217-9197 (toll-free).

Jane Zara
12-9-05

A handwritten signature in black ink, appearing to read "Jane Zara", with a stylized, cursive script.